For Debate . . .

Diverticular Disease of the Colon: A Deficiency Disease of Western Civilization

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We present a hypothesis as to the cause of diverticulosis coli which is consistent with its geographical distribution, its recent emergence as a medical problem, and its changing incidence. Diverticulosis appears to be a deficiency disease caused by the refining of carbohydrates which entails the removal of vegetable fibre from the diet. Consequently we consider it to be preventable.

Diverticulitis first became a clinical problem at the turn of the century, and the term "diverticulosis" first appeared in 1914. As recently as 1916 the disease was not important enough to merit a mention in textbooks.[1]

Though the present incidence of diverticulosis is unknown it is certainly endemic in our aged citizens. This dramatic increase in incidence occurred in only 70 years and cannot possibly be explained on a genetic basis. This change might be due to observer error and be apparent rather than real, but we believe that their writings show that the clinicians of the last century were just as capable as those of today of recognizing diverticulitis. We believe that there is another possibility—namely, that the colon's environment has changed and that diverticula are caused by the diet of so-called "civilized" countries.

Historical Impact of Diverticular Disease on Medicine

DIVERTICULA AS A CURIOSITY

The term "divertikel" was used by Fleischman in 1815.[2] Gross in 1845,[3] Cruveilhier in 1849,[4] Rokitansky in 1849,[5] Haberschon in 1857,[6] and Klebs in 1869[7] realized that diverticula were acquired and thought they were caused by constipation. The danger of diverticula as sites of infection and perforation was pointed out by Cruveilhier;[4] in 1859 Sidney Jones described vesicocolic fistula due to diverticulitis.[8] Harrison Cripps in 1888 collected 63 enterovesical fistulae but believed that they were caused by ingested foreign bodies.[9] He emphasized that they were usually the result of "inflammatory mischief" and not of cancer, but he blamed only diverticulitis in the case of Jones.

Virchow in 1853 described perisigmoiditis,[10] while Loomis in 1870 recorded peritonitis resulting from diverticulitis.[11] Since this complication was still regarded as a surgical curiosity 30 years later it is unlikely that perforated diverticulitis was common at that time.

Our nineteenth-century predecessors described diverticula and their complications accurately, but they regarded them as curiosities. Their concept of the pathogenesis of diverticula was surprisingly correct, and not until a century later, when cineradiography and pressure recording became available, were diverticula shown to be the result of functional obstruction due to segmentation dividing the colon into "little bladders." These become "trabeculated," with the colonic muscles thrown into ridges of varying thickness before the herniation of the mucosa takes place.[12-17]

Modern workers have only confirmed what Gross[3] believed—namely, that diverticula were caused by obstruction "by which the muscular fibres are separated from each other so as to permit the mucous membranes to protrude,. . . " Haberschon[6] blamed constipation for muscle thickening and for diverticula. Lane in 1885 realized that diverticula were not caused by distension, but that they were produced similarly to bladder diverticula—namely, by muscular contraction.[18] Bristowe in 1854 believed that diverticula were caused in a manner which resembled that operating in the secculated bladder and that costiveness might mimic the effects of obstruction.[19]

Surely, these men who, over a century ago, could foretell the findings of recent research would have recognized diverticulitis had it been common in their day.

EMERGENCE OF DIVERTICULITIS AS A CLINICAL PROBLEM

Graser in 1899 emphasized that diverticulitis led to perisigmoiditis and perforation,[20] and this warning was proved true within a decade. Five years later Beer described 18 infected diverticula and stressed that they could cause peritonitis, adhesions, fistula, and stenosis, but he still believed that they seldom caused symptoms.[21] "Diagnoses" such as pericolitis sinistra, perisigmoiditis, torsion and inflammation of appendices epiploicae lingered on, being still considered respectable by Bland-Sutton,[22] d'Arcy Power,[23] Donaldson,[24] and Lloyd Roberts.[25] Even in 1910 Gordon Taylor and Lakin[26] were reluctant to attribute peritonitis to diverticulitis, while Moynihan[27 28] and Mayo *et al.*[29] considered that diverticulitis mimicking cancer was still newsworthy at this time.

Dr. Telling, of Leeds, first saw the disease in 1899 when no one was familiar with it, but by 1908 he could describe all its complications, and in 1917 he published his classic description of diverticular disease. Even so, the condition was still not mentioned in textbooks in 1920.[1 30 31]

Diverticulitis surprised even surgeons of repute early in the century, but by 1920 Sir John Bland-Sutton remarked that "in the last ten years, acute diverticulitis is recognised with the same certainty as appendicitis and is a newly discovered bane of elders."[32]

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DIVERTICULOSIS AS A PROBLEM OF INCREASING MAGNITUDE

The advent of contrast radiology[33-37] showed diverticulosis to be common and prompted attempts to discover its true incidence. Necropsies showed that diverticula were rare before the age of 40. Necropsy and barium enema estimates of the prevalence of diverticula are given in Tables I and II. Neither method gives the true incidence. Necropsy series include more older subjects, and barium enemas are usually undertaken for symptoms. Radiological series exaggerate the incidence, while necropsy findings vary with the pathologists' interest in diverticulosis. The true incidence of diverticulosis could be found only by subjecting a sample of the population to annual barium enemas, but this is not practicable.

Bearing these limitations in mind, Mayo in 1930 was probably very near to the truth when he estimated that 5% of colons in patients over 40 bore diverticula,[38] as this figure agrees with contemporary necropsy series in the United States and Britain. Barium enemas were used more sparingly in those days but gave a similar incidence of 6-10%, which appears not to have increased greatly in the next 10 years. Morton, reviewing American necropsies,[39] and Edwards, quoting English barium enema studies,[40] recorded similar figures.

Recent studies from the U.S.A., U.K., Australia, and France indicate that the incidence of diverticulosis has risen dramatically. Between one-third and one-half of those over 40 have diverticula, and the incidence rises to two-thirds at 80 years.[41-42] The disease must have become commoner in Australia, as it was much less frequent there in the 1940s.[43]

Diverticular disease was almost unknown in 1900, *but has become the commonest affliction of the colon in Western countries within* 70 *years, the traditional life span of man.*

Geographical Distribution of Diverticular Disease

This dramatic rise in the incidence of diverticulosis has occurred only in economically developed countries, whose diet has changed only recently in the time scale of human nutrition. There are few diseases whose incidence varies so much throughout the world as that of diverticulosis—namely, from nil to nearly 30% of the population. A close relation exists between this incidence and economic development. The incidence in each developing country varies with the state of their economic development and corresponds to that which pertained in Western countries at a similar time in their industrial development when their pattern of life was similar. Obviously due allowance has to be made for the fact that diverticular disease takes about 40 years to develop, even after the colon's environment has been changed. Therefore diverticular disease would not be expected to be seen in a community until it had departed from its traditional eating habits for half a life-time. Consequently even where the incidence may one day be great the disease cannot be expected to be common until this unfavourable environment has operated for many years. Thus the condition would be expected to be almost unknown in sub-Saharan Africa to this day, and this is indeed the case. *Epidemiological evidence indicates precisely this relationship.*

The greatest contrast is to be seen between the Western world, including North American Negroes, and rural Africa

TABLE I—*Incidence of Colonic Diverticula: Necropsy Series*

Country and Author	Incidence of Diverticula		No. in Series	Comments
	%	No.		
United Kingdom:				
Drummond (1917)[59]	4·4	22	500	
Fifield (1927)[66]	2·1	—	10,167	London Hospital, 55% of subjects were under age 30, so incidence over 40 would probably have been 5%
Parks (1968)[41]	37	111	300	Northern Ireland, 50% in ninth decade
United States:				
Hartwell and Cecil (1910)[11]	5	—	81	New York, 1909-10
Rankin and Brown (1930)[67]	5·6	111	1,925	All but one subject aged over 40
Oschner and Bargen (1935)[78]	6·9	—	447	All necropsies in one year
Kocour (1937)[64]	3·58	—	7,000	Over age 40
	15·2	—		White women over age 70
	7·1	—		White men over age 70
	2·0	—		Coloured man over age 70
	3·0	—		Coloured woman over age 70
Morton (1946)[39]	6·3	—	8,500	Rochester, N.Y.
Australia:				
Cleland (1968)[43]	2·6	78	3,000	Refers to 1940-8. Indicence rose with age
	6·2	36	589	Over age 70 in this period
Hughes (1969)[42]	45	90	200	43% if caecal diverticula excluded

TABLE II—*Incidence of Colonic Diverticula: Barium Enema Series*

Country and Author	Incidence of Diverticula		No. in Series	Comments
	%	No.		
United Kingdom:				
Spriggs (1920)[68]	0·6	6	1,000	
Spriggs and Marxer (1925)[98]	10	100	1,000	Mainly adult patients. Importance of postevacuation film had been realized
Edwards (1934)[12]	10·8	—	507	Period 1925-31, King's College Hospital. Patients aged over 40
Grout (1949)[89]	8	—	2,179	
Edwards (1953)[40]	16	25	1,623	Relates to previous 13½ years and all patients over age 35
Manoussos *et al.* (1967)[90]	7·6	—	109	7·6% below age 60) Deliberate study by Ba meal and
	34·9	—		34·9% over age 60) follow-through in normal people
Sweden:				
Lunding (1935)[91]	4·2	87	2,090	
France:				
Debray *et al.* (1961)[92]	40	—	500	40% over age 70. All patients had gastrointestinal symptoms
United States:				
Enfield (1924)[93]	1·2	—	—	Found incidentally on barium studies
Mayo (1930)[38]	5·71	1,819	31,838	Mayo Clinic
Rankin and Brown (1930)[67]	5·67	1,398	24,620	Mayo Clinic
Oschner and Bargen (1935)[78]	7	—	2,747	Enemas given for intestinal symptoms
Willard and Bockus (1936)[79]	8·2	38	463	Consecutive enemas in private practice
Eggers (1941)[94]	7·5	—	647	Barium meal followed through colon
	44·5	—	428	Barium enemas
Allen (1953)[95]	30	—	2,000	Enemas given for symptoms. No diverticula under age 35, 5% at 45, 66% in older age groups
Welch *et al.* (1953)[96]	8·5	—	47,000	Collected series; 66% incidence over age 80
Smith and Christensen (1959)[97]	22	—	1,016	Consecutive enemas. Years 1954-8; incidence doubled at 80 years

and Asia. Countries which have only recently become industrialized have an intermediate incidence; highly industrialized Sweden has three times the incidence of Finland.[44]

DIVERTICULAR DISEASE IN AFRICA

Africa was least affected by the economic changes that occurred elsewhere just over a century ago, and our studies emphasize the almost total absence of diverticular disease in the rural Africans. During 20 years of practising surgery in Africa, mostly in a teaching hospital, one of us (D.P.B.) encountered not a single case of diverticulitis, nor did his surgical colleagues see the condition.

In the 2,000-bed Baragwanath Hospital, Johannesburg, serving the most urbanized Africans, Keeley[45] found no diverticula in 2,367 necropsies from 1954 to 1956, and Solomon[46] saw none in 600 barium enema examinations. The disease is extremely rare in the Bantu.[47]

At Mulago Hospital, Kampala, Davies found only two diverticula in 4,000 necropsies in 15 years.[48] Hutt[49] saw none in eight years in his pathological department, while Templeton[50] found only three caecal diverticula in 300 colons carefully scrutinized at necropsy. In Nairobi Miller[51] saw diverticulitis once in the past 11 years, and Chapman[52] saw one case in Durban in 14 years. Jain[53] served nine years in the Congo and Badoe[54] 16 years in Ghana, but both saw diverticulitis only once. Admittedly, diverticulosis might be missed where facilities are limited, but it is inconceivable that diverticulitis would be unrecognized consistently at major African hospitals with first-class staff and equipment and extensive necropsy experience. As the disease is so rare, even in African teaching hospitals, it is not surprising that 37 questionnaires completed recently by doctors serving in rural African hospitals showed that they had seen only two cases of diverticular disease between them, though some of them had had over 20 years' experience in Africa.

DIVERTICULAR DISEASE IN OTHER DEVELOPING COUNTRIES

The disease is almost as rare in Asians as it is in rural Africa. Tinckler saw 10 cases in the one and a half million Chinese, Indian, and Malay inhabitants of Singapore in five years, but three in only 15,000 Europeans.[55] Kutty,[56] in three years at Kuala Lumpur, found no diverticula at necropsy and de Beaux saw only a single case among 137,000 native Fijians as against two in the 7,500 resident Europeans.[55] Saidi,[57] Anderson,[58] and Wright,[59] did not see the disease in Shiraz (Iran), India, and New Guinea respectively. Kim found no diverticula in 500 necropsies in Korea.[60]

We know that these countries have fewer elderly people than do the industrial countries, but this could not affect the issue sufficiently to account for the almost complete absence of the disease in developing countries.

This survey confirms that diverticular disease is rarely found in peoples whose eating habits have changed but little up to the present.

In theory the amount of fibre in a diet may be reduced not only by the refining of flour and cereals but also by the substituting of refined sugar for unrefined foodstuffs that were previously eaten. In practice these processes occur together, as both follow industrialization.

In Britain the diet changed radically around the year 1880, though stone-ground white flour had been freely available since 1800.[61] A daily diet of 21 oz (600 g) of stone-ground wheat mixed with rye together with oatmeal porridge was not uncommon as late as 1860. Much of the fibre had been removed by stone grinding, but in 1880 the introduction of roller milling removed two-thirds of the remaining fibre from the flour. Concurrently, increasing prosperity, improvements in rail and sea transport, and refrigeration made other foods cheap and available. Meat imports doubled, and refined sugar and jam (which is 60% sugar) became part of the diet of the poorest classes.[62] The consumption of refined sugar almost doubled between 1865 and 1890,[63] and this was accompanied by a fall in the consumption of bread; this trend has continued to the present except during the two world wars.

If this swing from a high-residue diet to a low-residue diet is responsible for diverticulosis then the disease would be expected to be prevalent about 40 years after 1880, and, in fact, diverticulosis was common in Britain by 1920.

Likewise, increased prosperity led to a fall in the consumption of maize by the American Negro in this century. Formerly he was less prone to diverticulosis than his white compatriots,[64] but this difference has almost disappeared.[63] The raised incidence of diverticulosis in Japanese who have migrated to Hawaii compared with those who stayed in Japan has accompanied similar dietary changes.[65]

We believe that around 1880 the British diet was depleted of fibre sufficiently to damage the colon. The evidence suggests that the refining of flour and other cereals is the primary cause of diverticulosis, while the consumption of refined sugar at the expense of bread further increased the loss of dietary fibre.

Close observation of the dietary changes that are taking place in sub-Saharan Africa, where the low-residue diet has not been eaten for long enough to cause diverticulosis, is necessary. It has been postulated that appendicitis is caused by eating refined carbohydrates,[63 66 67] and if this and our hypothesis are correct then appendicitis, which is now becoming increasingly common in educated Africans, will be followed by the appearance of diverticulosis after a time interval similar to that which separated their recognition in Britain.

Postulated Relation

If a low-residue diet causes diverticulosis this must be related to colonic segmentation, which is the mechanism responsible for mucosal herniation. The role of segmentation in colonic physiology and in the genesis of diverticula is summarized in Fig. 1.[14-16 68]

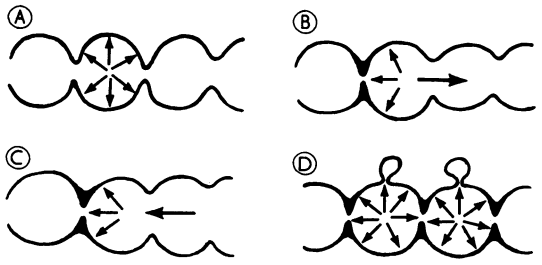

FIG. 1—Role of segmentation in colonic physiology and the pathogenesis of diverticula. Segmentation is concerned in the transportation and halting of faeces in the sigmoid colon. Diagram A shows a segmented colon; one segment has produced pressure by contracting. Diagram B shows how relaxation of the contraction ring on one side of this segment allows its contents to move into the next segment which harbours a lower pressure; this is the mechanism by which contents are moved. Diagram C shows how faeces are halted; contraction rings act as baffles which slow and finally halt contents, and a pressure change results. Segmentation is seen in the sigmoid as faeces are shunted back and forth. Diagram D shows a segmented colon acting as a series of "little bladders" whose outflow is obstructed at both ends and which extrude diverticula by generating high localized intrasegmental pressures. Segmentation is essential to the pathogenesis of diverticula. Any factor that causes segmentation to occur more frequently or more efficiently favours the causation of diverticular disease. (Reproduced by kind permission from *Annals of the Royal College of Surgeons of England.*)

We suggest that an unrefined diet containing adequate fibre may prevent diverticulosis for the following reasons:

(1) The colon that copes with a large volume of faeces is of a wide diameter and does not develop diverticula.[69 70] Such a colon, having a wide bore ab initio, segments less efficiently than does a narrow colon and is less prone to diverticulosis.[16]

(2) In most instances the food residue passes through the African's gut within 48 hours,[71] whereas in an Englishman this may take more than twice as long.[72 73] Thus the African's colon absorbs water for less time and has to propel a less viscous faecal stream. Hence the African's colon probably produces less pressure, and is less apt to become "trabeculated" and to bear diverticula.

(3) In Western countries custom often demands the suppression of the call to stool; this favours drying of the faeces and increased pressure generation. On the other hand the South African Bantu passes large moist motions without straining.[71]

In short, the swiftly passed soft stool subjects the sigmoid to less strain and does not favour the development of diverticula.

Effects of High-residue Diet on Established Disease

If a lack of fibre causes diverticulosis then the symptoms of diverticular disease might be alleviated by replacing the fibre, in the form of bran, in the diet. This has proved to be the case in a trial of 70 patients with the disease. Bran has relieved or abolished abdominal aching and pain and distension in over 80% of patients. Even severe colic which first was diagnosed as left renal colic disappeared on a high-residue diet. It is not yet known whether this diet prevents diverticulitis, but the symptoms of painful diverticular disease are usually diminished or abolished by adding bran to the diet.[74]

Origin of Low-residue Diet

Previous regimens of treatment were founded on a misunderstanding of the cause of diverticulosis. Spriggs and Marxer in 1927 believed that constipation and faeces stagnating in the sigmoid led to infection, so they gave paraffin to cleanse the colon.[75] Undigested fragments of food and bone associated with diverticulitis were reported by Cripps,[9] Bland-Sutton,[22] and Brewer,[76] and were believed to cause perforation. Hence a low-residue diet was advocated by authorities such as Slesinger,[77] Oschner and Bargen,[78] Willard and Bockus,[79] Brown and Marcley,[80] and Edwards.[81]

For nearly 50 years the low-residue diet has been the mainstay of medical treatment despite the lack of any evidence that it is of benefit. We believe that this diet is contraindicated in patients who suffer from diverticular disease because it is the cause of the condition.

Diseases Associated with Diverticulosis

The epidemiology of colon diverticula is closely linked with other non-infective diseases of the bowel. Communities free from the condition are equally exempt from benign polyps of the colon and have a very low incidence of colonic cancer and ulcerative colitis.[82] The association of gall-bladder disease, hiatus hernia, and diverticulosis is sometimes called "Saint's triad", but diverticula are also commonly associated with cardiovascular disease, duodenal ulcer and appendicitis,[83 84] and with diabetus mellitus.[85]

All these diseases are rare in people who have adhered to their traditional diet, and Cleave *et al.*[63] used the term "saccharine disease" (pronounced like the River Rhine) to include these diseases which they believe are caused by an excessive consumption of refined carbohydrate. If this is true these diseases should become commoner in the developing countries as they change to a Western diet.

Conclusion

Historical and epidemiological studies provide abundant evidence that diverticulosis coli is a disease of economically developed nations who eat a diet in which the carbohydrate is refined. Feeding rats with high-residue and low-residue diets, and a clinical trial in which a high-residue diet was given to patients with the disease, also indicate a cause-and-effect relationship between a low-residue diet and diverticular disease.

The disease and its complications are an increasing problem in Western countries, and we believe that much greater attention should be paid to preventive measures. It is worth remembering that the rise in the death rate from diverticular disease was halted in Britain only during the war and immediate postwar years, when white bread was not available and refined sugar was strictly rationed (Fig. 2). Perhaps a return to this high-residue diet would achieve more than all our surgical endeavours.

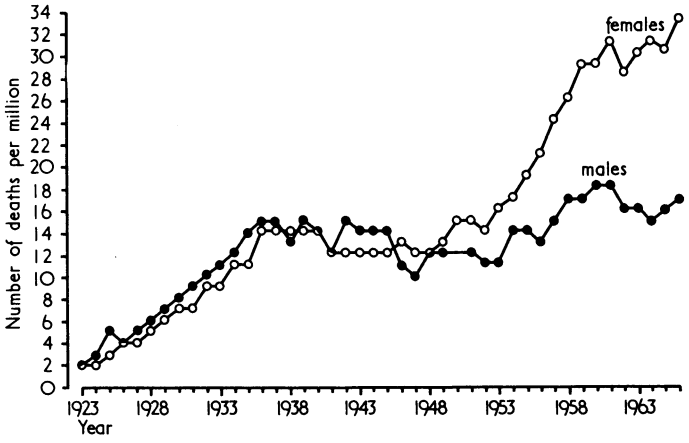

FIG. 2—Crude death rate for diverticular disease: Registrar General's statistical review of England and Wales 1923-66. (From Cleave, Campbell, and Painter.[63])

Once these causative factors have been recognized it is the duty of the profession to point the way to prevention, even if it entails issuing a warning with regard to such popular foodstuffs as white flour, both brown and white sugar, confectionery, and foods or drinks which contain unnaturally concentrated carbohydrates. Diverticular disease is a deficiency disease and, like scurvy, it should be avoidable. By retracing our dietary steps it should be possible to prevent its appearance in future generations and perhaps to lessen the incidence of carcinoma of the colon which has a similar epidemiology.

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General Practice Observed

Expressions of Morbidity in General Practice

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Morbidity studies in general practice measure the impact of disease on the primary physician and may or may not reflect the prevalence of disease in the community. A variety of studies have been conducted in the past two decades focusing on different aspects of this problem. Logan and Cushion[1] studied the whole spectrum of morbidity in general practice; Fry[2] concentrated on the natural history of specific diseases; Shepherd *et al.*[3] focused on psychiatric morbidity. These and other workers have related consultation rates and observed prevalence rates to a variety of characteristics such as the age, sex, and social class of patients at risk. Fry, by studying patients over a period of time, has been able to relate his findings to indices such as fatality rates and other measures of disability and to the use of resources.

To obtain a deeper understanding of the impact of illness on the general practitioner this study was designed to supplement the usual measurements of consultation rates and observed prevalence rates with measurements of the time devoted to the management of different diseases, the disability they produce, the diagnostic activity they provoke, and the extent to which the management of illness may be delegated to paramedical workers.

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Practice

The study took place in a three-man partnership practice providing medical care for 4,455 patients. Each of the doctors